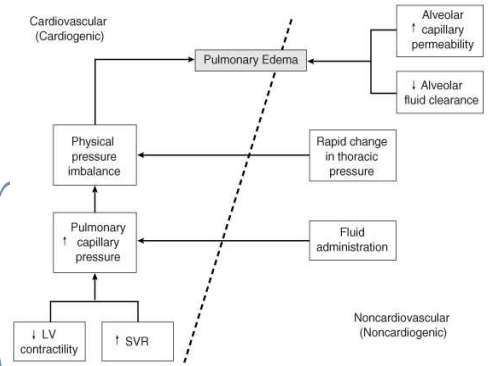
non-cardiogenic causes of pulmonary oedema

general

- The most common cause of noncardiovascular pulmonary edema is ARDS.
- In most syndromes of noncardiovascular pulmonary edema a combination of factors including inflammation, direct damage to the capillary-alveolar membrane, and hypoxia causing leakage of the capillary-alveolar barrier and decreased alveolar fluid clearance are prominent pathogenetic mechanisms

pathophysiology of cardiogenic vs non-cardiogenic pulmonary oedema



fluid movement = k[(Pc + OSMi) - (Pi + OSMc)]

causes

Increased Alveolar-Capillary Permeability and Reduced Alveolar Fluid Clearance
Acute respiratory distress syndrome
Neurogenic pulmonary edema
Preeclampsia
Drug Substance and Toxic Inhalation Pulmonary Edema
Opiate overdose
Anticancer therapy
Salicylate overdose
Thiazolidinedione-related pulmonary edema
Radiocontrast-related pulmonary edema
Environmental and toxic inhalation pulmonary edema
Other drugs[35]: tricyclic antidepressants, hydrochlorothiazide[R]
Alveolar-Capillary Pressure Imbalance
Perioperative pulmonary edema
Elevated Capillary Pressure: Excessive Fluid Transfusion or Fluid Shifts
Peripartum pulmonary edema
Ovarian hyperstimulation syndrome[13]
Exertional pulmonary edema
Hypoxia-Related Pulmonary Edema
High-altitude pulmonary edema
Rapid Change in Thoracic Pressure
Post upper airways obstruction
Post pneumonectomy
Post evacuation of pleural effusion
Post evacuation of pericardial fluid (rare)

drug, substance & toxic inhalation

- The most common cause of drug-induced pulmonary edema is the use of cardiodepressants such as beta-adrenergic blockers and some calcium blockers and antiarrhythmics.
- Pulmonary edema has been associated with the intake or toxicities of drugs that provoke edema through different mechanisms.

postoperative

- a relatively common finding, especially when the surgical procedures are extensive and the patient is elderly and suffers from significant cardiac comorbidities.
- The pathogenesis of postoperative pulmonary edema is diverse. Cardiovascular factors are major contributors to pulmonary edema in patients sustaining a postoperative myocardial infarction, whereas alveolar capillary leakage and reduced alveolar fluid clearance play a major role in postoperative pulmonary edema related to major infections.
- In the absence of predisposing risk factors, fluid overload may be the main reason for pulmonary edema.

postpneumonectomy

- Pulmonary edema develops in 2.5% to 4.5% of patients after pneumonectomy.
- The pathogenesis of this syndrome is unknown. However, a combination of large fluid transfusion, excessive negative pressure in the operated hemithorax due to underwater suction systems, major lymphatic interruption related to extensive surgery, and damage to the alveolar-capillary membrane have been implicated as possible causes.
- The fatality of this syndrome is significant, up to 85% in one series. Hence, prevention is of great importance.

pregnancy-related

General:
- Pulmonary edema is an uncommon complication of pregnancy.
- It usually occurs in the peripartum period from a combination of factors, including mobilization of fluids and fluid administration, use of tocolytic treatment, and preeclampsia.

Tocolytics:
- Tocolytic treatment use is the most common cause of pregnancy-related pulmonary edema (26%). In most cases, multiple tocolytics that include a beta-mimetic agent are administered, probably inducing a significant increase in systemic vascular resistance.

Cardiac disease
- In a further 26%, pulmonary edema is related to a preexisting cardiac disease that is exacerbated during the peripartum period and in combination with the large volume shifts during this period that induce pulmonary edema.

Fluid overload
- Fluid overload per se is the main etiology of pulmonary edema in 22% of patients. In these cases pulmonary edema is related to a large volume transfusion of approximately 6 L over a short period of time.

Preeclampsia
- Finally, preeclampsia is the main cause of pulmonary edema in 18% of cases. Preeclampsia causes pulmonary edema through a combination of cardiovascular (reduced left ventricular contractility and increased systemic vascular resistance) as well as noncardiovascular factors (endothelial damage leading to increased fluid leak into the alveoli).

Peripartum cardiomyopathy
- Peripartum cardiomyopathy is an important entity causing pulmonary edema but should be listed among the causes of cardiovascular pulmonary edema.

postobstructive

- This syndrome occurs after the relief of either acute or chronic obstructions of the upper airways.
- The most common cause is relief of obstruction occurring during anesthesia, although other acute causes of upper airways obstruction such as epiglottitis, croup, foreign bodies, strangulation, tumors, goiter, vocal cord paralysis, and obstruction of endotracheal tubes have been reported.
- Pulmonary edema develops minutes to hours after the relief of obstructions, and its incidence may be up to 10% after relief of acute obstructions and up to 40% after relief of chronic obstruction
- The pathophysiology of postobstructive pulmonary edema is not known, but a combination of increased pulmonary capillary pressure owing to significant negative pressure during the obstructive period combined with hypoxia leading to decreased alveolar fluid clearance, increased systemic vascular resistance due to sympathetic overflow, and stress failure of the alveolar-capillary membrane have all been postulated as possible causative mechanisms.

re-expansion

- Pulmonary edema may occur after evacuation of a large pneumothorax or pleural effusion.
- The pathogenesis is not known, but leaks in the alveolar capillary membrane after prolonged atelectasis and rapid re-expansion were suggested by some authors. The treatment is symptomatic.
- The prognosis is not known, although in some older series a mortality of up to 20% was described.

neurogenic

- an uncommon complication of various neurologic insults such as head injury, intracranial and subarachnoid hemorrhage, as well as some acute neurologic diseases, including seizures, tumors, hydrocephalus, and neurosurgical procedures.
- The clinical course is highly variable.
- The syndrome is often acute and fulminant but may be subclinical and smoldering, manifesting as a mild progressive shortness of breath.
- The pathogenesis of neurogenic pulmonary edema is probably related to a combination of increased sympathetic discharge leading to both increased systemic vascular resistance and decreased left ventricular contractility, as well as increased alveolar-capillary leakage.
- some authorities in the neurologic literature advocate the use of alpha-adrenergic blockers such as phentolamine and phenoxybenzamine owing to the possible role of sympathetic overflow in this syndrome. However, these treatment options have never been examined in a prospective, controlled study and hence should be used with caution.

high-altitude

- a syndrome related to climbing to high altitudes.
- Its exact pathophysiology is unknown, but in a few recent studies it was demonstrated that a combination of hypoxia-induced vasoconstriction leading to increased pulmonary capillary pressure and alveolar fluid transudation together with decreased alveolar fluid clearance leads to alveolar fluid accumulation and edema.
- The clinical presentation is typical, ranging from cough to full-blown respiratory failure.
- The treatment includes oxygen administration, rapid descent to lower altitudes or simulated descent by a hyperbaric chamber, and possibly administration of calcium antagonists. Furosemide and dexamethasone are probably not efficacious.

exertional

- Pulmonary edema has been reported to occur during and especially after strenuous exercise and diverse sports activities and is especially common after prolonged swimming in cold water.